

- 18 Barclay CS, French MAH, Ross LD, Sokol RJ. Successful pregnancy following steroid therapy and plasma exchange in a woman with anti-Ro (SS-A) antibodies. Case report. *Br J Obstet Gynaecol* 1987;94:369-71.
- 19 Carpenter RJ, Strasburger JF, Garson A, Smith RT, Deter RL, Engelhardt HT. Fetal ventricular pacing for hydrops secondary to complete atrioventricular block. *J Am Coll Cardiol* 1986;8:1434-6.
- 20 Sholler GF, Walsh EP. Congenital complete heart block in patients without anatomic cardiac defects. *Am Heart J* 1989;118:1193-8.
- 21 Michaelsson M, Engle MA. Congenital complete heart block: an international study of the natural history. *Cardiovasc Clin* 1972;4:85-101.
- 22 Dewey RC, Capeless MA, Levy AM. Use of ambulatory electrocardiographic monitoring to identify high-risk patients with congenital complete heart block. *N Engl J Med* 1987;14:835-9.
- 23 Pinskey WM, Gillette PC, Garson A, McNamara DG. Diagnosis, management, and long-term results of patients with congenital complete atrioventricular block. *Pediatrics* 1982;69:728-33.
- 24 Karpawich PP, Gillette PC, Garson A, Hesslein PS, Porter C-B, Macnamara DG. Congenital complete atrioventricular block: clinical and electrophysiologic predictors of need for pacemaker insertion. *Am J Cardiol* 1981;48:1098-102.
- 25 Esscher E, Michaelsson M. Q-T interval in congenital complete heart block. *Pediatr Cardiol* 1983;4:121-4.

ABSTRACTS IN CARDIOLOGY

Rheumatic fever: pathogenesis revealed

Generations of medical students have been taught concepts about the pathogenesis of rheumatic fever which have great appeal. The concept of "molecular mimicry" lays down that certain components of the wall of certain strains of streptococci fortuitously show close molecular similarity with connective tissue components of heart valves and myocardium and that antibodies produced against the bacterial antigen cross react against the heart, causing damage. The theory always had

inconsistencies. The Aschoff body has no histological features that suggest humoral damage and bound gamma globulin is not present in the tissues. The present study shows that T cells from the hearts of subjects who have had rheumatic fever can recognise both cardiac and bacterial wall antigens and the damage to the heart is probably cell based—a small but important change in thinking

M J DAVIES

Human heart-infiltrating T-cell clones from rheumatic heart disease patients recognize both streptococcal and cardiac proteins

L Guilherme, E Cunha-Neto, V Coelho, R Snitcowsky, P M A Pomerantzeff, R V Assis, F Pedra, J Neumann, A Goldberg, M E Patarroyo, F Pileggi, J Kahil

Background— β -Hemolytic streptococcal infection in developing countries still causes thousands of cases of rheumatic heart disease, demanding surgical valve correction. Antigenic mimicry between self and streptococcal components has been proposed as the triggering factor leading to autoimmunity in individuals with genetic susceptibility. Although heart streptococcal-M protein cross-reactive antibodies have been demonstrated, heart tissue damage seems to be T lymphocyte-dependent. We studied the infiltrating T lymphocytes in rheumatic heart lesions with the aim of understanding the role of cellular immune response at the site of the lesions.

Methods and results—We obtained 107 T-cell clones from surgical fragments of cardiac tissue from four rheumatic heart disease patients. We tested their capacity to recognize streptococcal M protein-derived synthetic peptides and heart pro-

teins. We found eight infiltrating T-cell clones from all four patients that simultaneously recognize streptococcal M and heart proteins. Among the M-proteins sequences tested, only synthetic peptides corresponding to regions 1 through 25, 81 through 103, and 163 through 177 were simultaneously recognized with heart protein fractions. Interestingly, regions 81 through 103 and 163 through 177 have been known to bear heart cross-reactive epitopes at the antibody level. Five of these clones are CD4⁺, and one is CD8⁺.

Conclusions—The presence of heart-M protein cross-reactive T-cell clones in rheumatic heart lesions suggests their direct involvement in the pathogenesis of this disease. The dissection of protective and pathogenic epitopes of streptococcal M protein is an important step in allowing the development of a safe anti-streptococcal synthetic vaccine. (*Circulation* 1995;92:415-20.)